



Endovascular Stent-graft Treatment for Stanford Type A Aortic Dissection

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Abstract *Objective:* The aim of the study is to summarise our experience of endovascular stent grafting for Stanford type A aortic dissection.

Design: Retrospective analysis at single centre.

Methods: From January 2001 to January 2009, we treated 45 cases of Stanford type A aortic dissection with endovascular stent grafting. The entry tear was located at the ascending aorta in 10 cases (DeBakey type I), the aortic arch in 14 cases and the distal aortic arch or proximal descending aorta in 21 cases in which the ascending aorta was also involved by the dissection. *Results:* The surgical success rate was 97.8% (44/45) and 30-day mortality rate was 6.7% (3/45). Type I endoleaks occurred in 10 cases: one patient died intra-operatively, four were successfully treated with ballooning, four were sealed with aortic cuffs and one case caused by left subclavian artery (LSA) reflux was sealed with an occluder. Average follow-up time was 35.5 ± 5.4 months. Up to the most recent review or death, 32 patients had complete thrombosis and 10 had partial thrombosis inside the false lumen. Two deaths occurred after 30-days postoperatively.

Conclusion: Endovascular stent-graft treatment is a minimally invasive and effective method to treat Stanford type A aortic dissection.

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According to the Stanford classification, all lesions dissections involving the ascending aorta are considered Stanford type A,¹ including DeBakey type I, II and III R, that is,

tearing retrograde to the arch and ascending aorta (AO).² Although traditional open aortic replacement is an effective treatment for Stanford type A aortic dissections (ADs),

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it is associated with a mortality rate ranging from 15% to 30%.^{3–6}

Though an endovascular approach has been proven useful for the management of abdominal aortic and thoracic aortic aneurysms and the treatment of Stanford type B dissections,^{7–10} it is only in the past decade that the endovascular approach has been examined for the management of type A dissections.^{11–17}

Since 2001, we have been treating Stanford type A ADs with endovascular stent-graft implantation. The purpose of this retrospective study is to present the results of our cumulative experience of the endovascular treatment of Stanford type A ADs.

Patients and Methods

Clinical data

From January 2001 to January 2009, 166 cases of Stanford type A ADs were treated at our hospital. Of these, 79 patients received open surgery, 42 patients declined surgical treatment and 45 were managed by endovascular stent grafting and included in this analysis. If the areas of the aortic valve and coronary artery ostia were not affected and the distance between the entry tear and the coronary ostia was >2.5 cm, patients were offered the option of endovascular repair. Patients were not offered endovascular management if the aforementioned criteria were not met, the dissection was trauma induced, or if the whole AO was affected. After discussion of the surgical options, the patient or their relatives made the decision as to the type of surgery.

There were 41 males and four females with a median age of 51 years (range, 38–79 years) included in this analysis. There were 30 cases of acute AD (≤ 14 days) and 15 cases of chronic AD (> 14 days). Computed tomographic angiography (CTA), magnetic resonance angiography (MRA) and aortic angiography were performed in all cases to determine the intimal entry tear location and the extent of the dissection. Preoperative echocardiography was performed to detect aortic valve insufficiency or regurgitation, coronary artery dissection and to measure the distance from the entry tear to the coronary artery ostium. Patient clinical data are presented in Table 1. Patients were categorised into three groups: group A ($n = 10$), DeBakey type I AD; group B ($n = 14$), entry tear located at the aortic arch; and group C ($n = 21$) entry tear at the distal aortic arch or descending aorta in which the ascending aorta was also involved by the dissection. All patients provided informed consent for the procedures and approval was obtained from the Institutional Review Board of our hospital.

Endovascular interventions

All patients underwent surgery under general anaesthesia without cardiopulmonary bypass. The stent grafts used included the Medtronic Talent™ (Medtronic Inc., Minneapolis, MN, USA), Cook Zenith™ (Cook Medical, Inc., Bloomington, IN, USA), Ancure II™ (XianJian Science and Technology Company, China) and Aegis™ (MicroPort Medical Co., Ltd, Shanghai, China). Stent diameters ranged from 30 to

42 mm and the length of the covered stent ranged from 100 to 150 mm. Aortic cuff diameters ranged from 32 to 42 mm and the length of the covered stent ranged from 50 to 80 mm. Stent grafts were oversized by 5–10%. The grafts for transposition of arch vessels were expanded polytetrafluoroethylene (ePTFE) Gore-Tex (W.L. Gore Associates Inc., Flagstaff, AZ, USA).

If the entry tear was located in the proximal descending aorta, the stent was placed near the ostium of the left subclavian artery (LSA) and if an endoleak occurred, the stent was expanded by balloon so that it was tightly adhered to the vessel wall. If the endoleak was caused by stent mobilisation or angulation, an aortic extending cuff was implanted. If the endoleak was caused by reflux from the LSA, an occluder was implanted through the left brachial artery to close the orifice of the LSA. In all cases, systolic blood pressure was controlled to approximately 90 mmHg during deployment of the prostheses. We did not use rapid pacing, cardiac arrest or over pacing. Representative images of group A, B and C patients are presented in Figs. 1–3. Patients were followed with CTA the first, sixth, and 12th month postoperatively, then yearly.

Group A

In nine patients aortic cuff stents ranging from 60 to 80 mm were used, and in one patient a 10-cm covered stent was used. In two patients, stents were introduced through the left common carotid artery (LCCA) and LSA–LCCA bypass was constructed prior to the stent implantation. In the other eight patients, stents were introduced through the right common femoral artery (RCFA). In two of these patients, the entry tears were close to the innominate artery such that sealing of entry tears with stents might have led to closure of the innominate artery, and possibly LCCA. Therefore, LSA–LCCA–right common carotid artery (RCCA) bypasses were performed. In all cases, the stent graft was placed under low blood pressure, placement was confirmed radiographically and was adjusted, if required, and then was released.

Group B

All patients were treated with supra-aortic transposition (Table 1) and one- or two-stage endovascular stent grafting. After the transposition procedure, stent grafts were implanted through the RCFA into the aortic arch in one or two stages to close the entry tears together with the ostia of supra-aortic great vessels. In two cases, the entry tears were close to the LSA and because the right vertebral artery (RVA) was not the dominant artery, revascularisation of the LSA was performed. In these patients, the ascending aorta was not affected since the tear was primarily on the aortic arch and thus had a normal length of ascending aorta for debranching.

Group C

The entry tear was located at the distal aortic arch in nine patients and at the descending aorta in 12 patients. A one-stage procedure was performed in all patients, and 17 were treated with endovascular stent grafting only. The LSAs in 10 patients were covered simultaneously, in whom four were shown to have RVA occlusion or stenosis $> 70\%$ by preoperative arteriography. LSA–LCCA bypass was

Table 1 Patient characteristics (N = 45).

	Group A: Entry Tear at Ascending Aorta	Group B: Entry Tear at Aortic Arch	Group C: Entry Tear at Descending Aorta or Distal Aortic Arch
N	10	14	21
Gender			
Male	9	13	19
Female	1	1	2
Age (y)	51 (41–71)	53 (38–75)	55 (41–79)
Dissection			
Acute	6	9	15
Chronic	4	5	6
Duration from onset to operation (d)	3–73	—	—
Acute chest pain	All	—	—
Sudden chest pain	—	All	All
Upper back pain	All	All	16
Hypertension	All	All	17
History of connective tissue disease	0	1 (Marfan syndrome)	0
Distance to coronary artery ostium (cm)	2.5–6	—	—
Distance to right innominate artery (cm)	0.5–4	—	—
Entry tear between BA and LCCA	—	4	—
Entry tear between LCCA and LSA	—	10	—
Entry tear at distal aortic arch	—	—	9
Entry tear at descending aorta	—	—	12
Diameter of intimal tear (cm)	0.8–2.0	0.8–1.6	0.9–1.5
Diameter of dissection (cm)	4.5–7.5	5.5–10.3	6–11.2
Left common carotid artery path	2	—	—
Right femoral artery path	2	—	—
Endovascular stent grafting and supra-aortic great vessel transposition	2	14	4
AO-LCCA and RCCA	—	1	—
AO-LSA and LCCA	—	3	—
LCCA-RCCA	—	8	—
LCCA-LSA	—	2	4
RCFA path	—	All	—
One-stage procedure	1	7	All
Two-stage procedure	1	7	—
Endovascular stent grafting only	8	—	17

Acute dissection defined as ≤ 14 days, chronic dissection defined as > 14 days.

Data are presented as number (range).

AO, ascending aorta; BA, brachiocephalic artery; LCCA, left common carotid artery; LSA, left subclavian artery; RCCA, right common carotid artery; RCFA, right common femoral artery.

performed to supply blood to the LSA. The remaining six patients did not receive LSA revascularisation because angiography indicated good blood supply of the RVA and Circle of Willis.

Results

The endovascular procedure was successfully performed in 44 of the 45 patients (97.8%). One intra-operative death occurred in group B. In this case, a bypass graft was placed between the LCCA and RCCA, and a stent graft was successfully deployed to seal the tear. A delayed type I endoleak was found, and balloon angioplasty was performed. During the procedure, the patient experienced cardiopulmonary arrest and died. In one case in group C,

the entry tear was located at the descending aorta, but the dissection involved the ascending aorta. Three hours after successful stent-graft implantation, the patient experienced a myocardial infarction and died. In group A, one patient died from gastrointestinal haemorrhage 25 days postoperatively. Thus, the 30-day postoperative mortality rate was 6.7% (3/45).

The mean follow-up of the 42 patients who survived beyond 30 days was 35.5 ± 5.4 months (range, 4–112 months). There were two deaths during the follow-up period; thus, the total mortality rate was 11.1% (5/45). One patient in group B with Marfan syndrome died 2 months postoperatively. The patient's family did not allow an autopsy; thus the cause of death is unknown. A patient with a DeBakey type I dissection was discharged and died from pneumonia and acute left ventricular failure 10 months postoperatively.

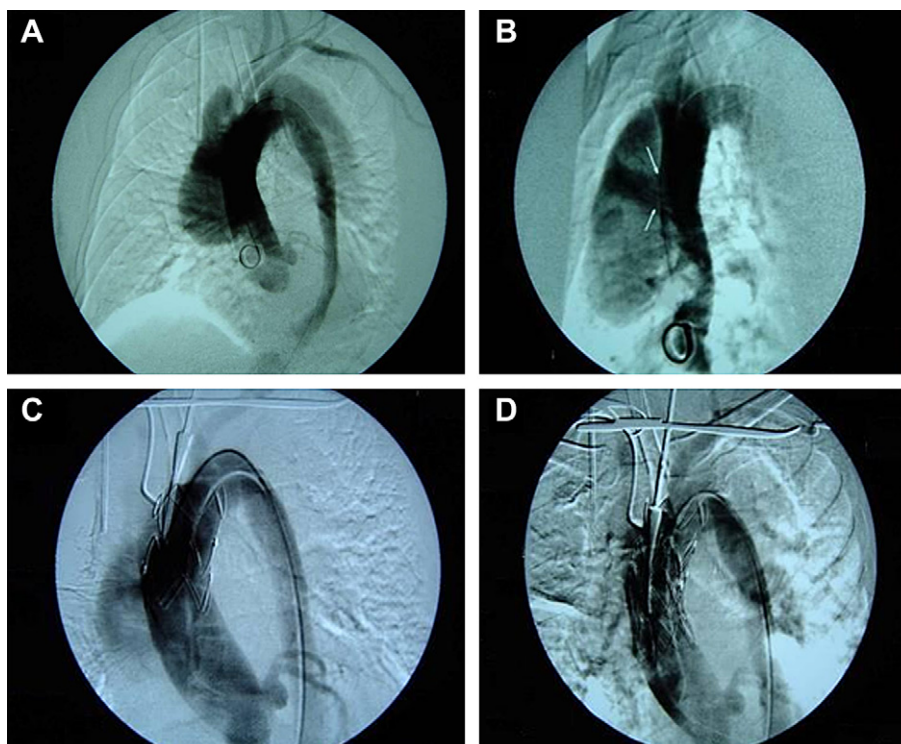


Figure 1 Group A. A) Preoperative DSA showed an aortic dissection with tear 1.5 cm from the right innominate artery, 5 cm from the ostium of coronary artery, and B) 2.0 cm in diameter. C) Endoleak remained after the first stent was implanted through LCCA. D) A second stent was implanted to close the tear and the leakage stopped.

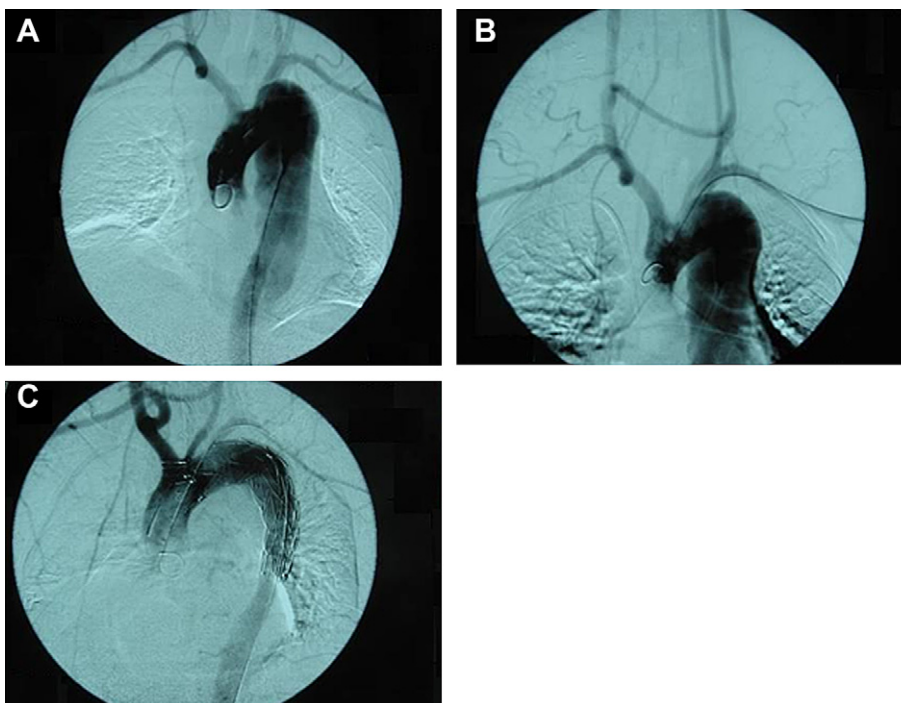


Figure 2 Group B. A) Tear on aortic arch between LCCA and LSA. Another tear was located below the level of the renal artery. B) Postoperative DSA showed good circulation in the carotid artery bypass. C) The right femoral artery was exposed, the aortic stent delivery system was inserted into brachiocephalic artery, the bare stent frame was placed to the left of the brachiocephalic artery, and the stent was released.

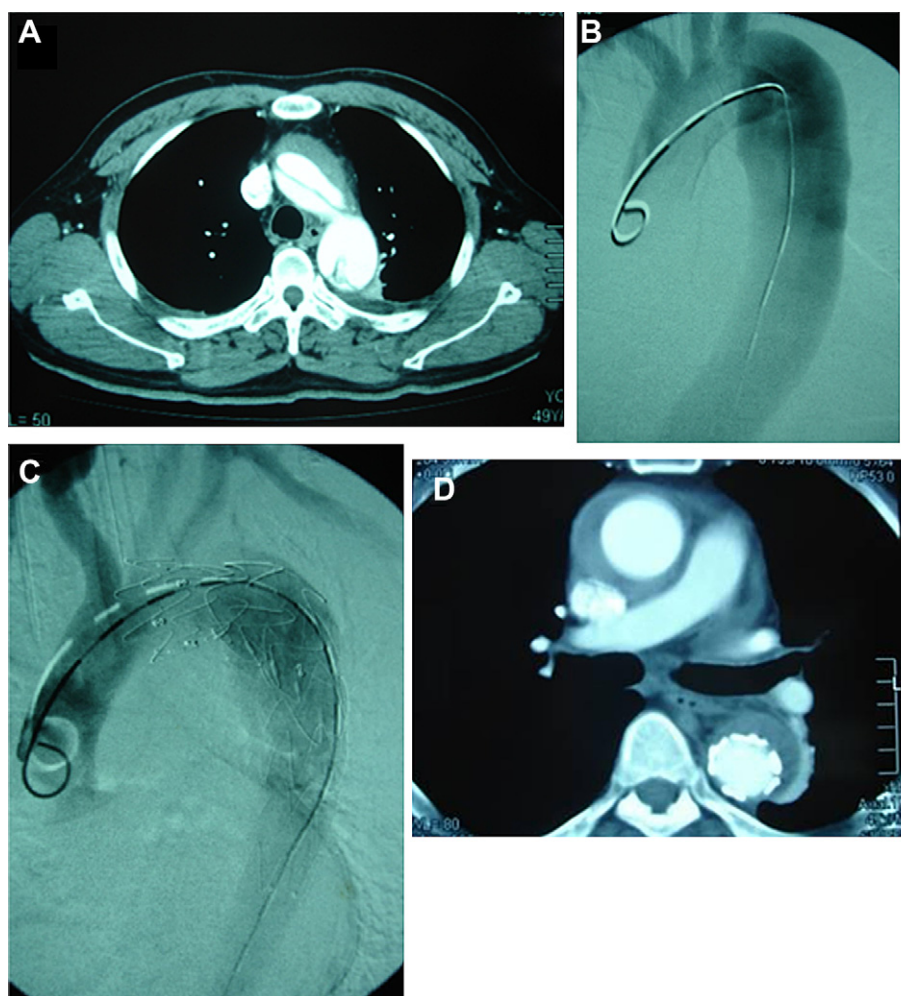


Figure 3 Group C. A) CTA revealed Stanford type A dissection. B) DSA showed tear at the distal LSA. C) Stent graft implanted. D) CTA 3 months postoperatively indicated the dissection is no longer present and thrombus formation inside the dissection cavity.

There was no evidence to suggest the death was related to the surgery. Up to the most recent follow-up or death, there were 32 cases with complete thrombosis and 10 cases with partial thrombosis inside the false lumen.

Type I endoleaks occurred in 10 patients (Table 2). The endoleak associated with the intra-operative death has been described above. In one case in which the LSA was occluded intra-operatively, the endoleak was considered to be caused by reflux of the LSA. An occluder was implanted to occlude the ostium of LSA. In four cases, the endoleaks

did not decrease after ballooning; however, they resolved after aortic extending cuff placement. In the other four patients, endoleaks were dramatically diminished after balloon expansion. No further treatment was performed. In group C, a secondary type II endoleak occurred 6 months postoperatively. It was assumed to be caused by reflux of the RVA, and no treatment was performed. During 2 years of follow-up, no significant change has occurred.

During the follow-up period, there were a total of three cases of cerebral infarction; thus the cerebral infarction

Table 2 Summary of complications by group.

	Group A: Entry Tear at Ascending Aorta	Group B: Entry Tear at Aortic Arch	Group C Entry Tear at Descending Aorta or Distal Aortic Arch
Mortality within 30-days postoperatively	1	1	1
Mortality after 30-days postoperatively	1	1	0
Type I endoleak	1	6	3
Cerebral infarction	2	1	0
Pseudo-aneurysm	1	0	0
Neck haematoma	0	1	0

rate was 6.8% (3/44, one intra-operative death was not counted). In group A, two patients who received endovascular treatment through the LCCA experienced infarctions at 3 weeks and 1 year postoperatively, respectively. In group B, one patient experienced an infarction on post-operative day 1. In all cases, studies of the revascularised branches revealed patency.

There were only two complications other than those previously described, and no patient experienced renal failure, pulmonary failure, myocardial infarction, deep vein thrombosis or paralysis. In addition, there were no cases of distal malperfusion, cardiac effusion or aortic regurgitation. In group A, one patient with DeBakey type I dissection had an LSA–LCCA–RCCA bypass with artificial grafts and stent implantation in a single-stage procedure. A pseudoaneurysm at the anastomotic site of the LSA and artificial graft was found 8 months postoperatively and a right axillary–right external iliac artery bypass with pseudoaneurysm resection was performed and the proximal ends of LSA and the artificial graft were ligated. A neck haematoma occurred in a patient in group B 3 weeks postoperatively and resulted in airway compression. The haematoma was evacuated and the patient recovered. Complications are summarised in Table 2.

Discussion

We have reported the use of an endovascular approach to treat 45 patients with Stanford type A ADs: the 30-day postoperative mortality rate was 6.7% (3/45) and with a mean follow-up of 35.5 ± 5.4 months, total mortality rate was 11.6% (5/43) and the cerebral infarction rate was 6.6% (3/45). The mortality rate reported for our series is at the low end of the reported surgical mortality of 7–30% for Stanford type A dissections.¹⁸ Despite the utility of the endovascular approach for Stanford type B dissections,¹⁹ experience of endovascular stent implantation for type A dissections is still somewhat limited. In the current analysis, we grouped patients based on the location of the entry tear.

Entry tear at the ascending aorta

In 2000, Dorros et al.²⁰ reported the first DeBakey type I case successfully treated with an endovascular technique. In 2002, we reported the first case of a DeBakey type I dissection treated with an endovascular technique in China²¹ and have since treated an additional 10 cases. The stents were implanted through the LCCA in two of the 10 cases described above. Because the early generation of the device was rigid, it was difficult to deploy the guide sheath through the aortic arch into the ascending aorta; thus initially the LCCA was chosen to facilitate the release of the stent. We first created a carotid shunt to build a LSA–LCCA bypass to assure the blood supply to the brain, then the LCCA was opened and the stent was inserted. In eight cases, we implanted stent grafts through the RCFA into the AO. Based on our experience, the RCFA path is suitable for patients with a lesser curvature of the aortic arch. Furthermore, the RCFA approach avoids blockage of common carotid arteries, the necessity of carotid artery

bypass and potentially the occurrence of cerebrovascular events.

Entry tear at the aortic arch

To treat Stanford type A dissections with the entry tear located at the aortic arch, protection of the supra-aortic great vessels to guarantee the blood supply to the head and neck after sealing the entry tear is necessary. In our experience, the landing area for the stent was not usually sufficient. In this situation, transposition of the aortic arch great vessels can provide enough landing area for endovascular stent implantation. Bergeron et al.²² reported transposition of all of the large arch vessels in 15 cases and partial vessel transposition in 10 cases prior to stent-graft implantation, with a surgical success rate of 92%. In our study, one patient with the entry tear at the aortic arch died from sudden cardiac arrest when a balloon was expanded to treat a type I endoleak. We hypothesise that over expansion of balloon lead to rupture of aneurysm and subsequent cardiac arrest; however, the patient's family did not agree to an autopsy, so the exact cause of death is uncertain.

Entry tear located at the descending aorta or distal aortic arch

Most surgeons use a hybrid technique to treat Stanford A dissections with the entry tear located at the descending aorta or distal aortic arch, which includes replacing the AO and aortic arch (semi-arch or total arch) with artificial vessels, and then stent implantation through the open aorta to block the intimal tear. For patients in whom the LSA needs to be closed, whether LSA revascularisation is necessary or not depends on the blood supply in the RVA and the left upper limb. Jazayeri et al.²³ reported the successful treatment of two Stanford type A dissections, one with the tear at the descending aorta and the other at the aortic arch. Both cases had ascending aorta replacement, with additional half aortic arch replacement in one case. In both cases, the stent graft was implanted inside the artery to block the tear on the descending aorta and aortic arch. Kato et al.²⁴ reported 10 cases of Stanford type A dissection successfully treated with stent-graft implantation, and Uchida et al.²⁵ reported a mortality rate of 4.6% and 5-year survival of 92.8% in 65 cases of acute Stanford type A dissection treated with aortic arch replacement plus stent-graft implantation to block the tear at distal aorta.

If the intimal entry tear at the distal aortic arch or proximal descending aorta is too close to the LSA, it may be necessary to cover the LSA to achieve a sufficient landing area for the stent, and it is debated whether revascularisation of the LSA is necessary. Based on our experience, the decision should be made based on the blood supply in the RVA.

Endoleaks

There were 10 type I endoleaks following stent implantation in our series (22.2%). All were treated with balloon expansion, and the endoleak was decreased in four

patients. We perform balloon dilatation only in the landing area of the stent graft in front of the entry tear, not for the whole length of the stent graft. The purpose is to make the stent adhere well to the blood vessel wall. The balloon dilatation time is very short, only 2–3 s. The systolic blood pressure is reduced to 90 mmHg during balloon dilatation to prevent shifting of the stent graft. Balloon dilatation is performed only once, and if the endoleak persists an aortic extending cuff is used. Our selectivity in choosing cases in which to do balloon dilatation is likely the reason no retrograde dissections occurred (i.e., we did not do balloon dilatation at the entry site tear or for the whole stent graft).

In cases in which an endoleak is not decreased by balloon expansion, reasons for the endoleak should be investigated. If the edge of the stent was close to the entry tear which was not completely sealed, an aortic extending cuff can be implanted to seal the tear. If the endoleak is caused by reflux from the sealed LSA, an occluder can be placed to close the ostium of LSA.

In conclusion, endovascular stent-graft treatment is a minimally invasive and effective method to treat Stanford type A AD. When necessary, it can be combined with the arch vessel bypass to obtain sufficient anchoring area for lesions involving the aortic arch to assure blood supply to the head and neck.

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Conflict of Interest

None.

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